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FILE 'HOME' ENTERED AT 13:41:57 ON 24 JUN 2000

=> s (Valpha20 and Valpha22) (B) (TCR)

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FILE 'BIOSIS' ENTERED AT 13:42:52 ON 24 JUN 2000
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=> s (Valpha20 and Jalpha22) (P) (TCR)

PROXIMITY OPERATION NOT ALLOWED
PROXIMITY OPERATION NOT ALLOWED
PROXIMITY OPERATION NOT ALLOWED
PROXIMITY OPERATION NOT ALLOWED

Certain operators may not be nested in combination with other operators. A nested operator is valid only when it occurs at the same level or above the operator outside the nested phrase as determined by the following precedence list:

1. Numeric
2. (W), (NOTW), (A), (NOTA)
3. (S), (NOTS)
4. (P), (NOTP)
5. (L), (NOTL)
6. AND, NOT
7. OR

For example, '(MONOCLONAL(W)ANTIBOD?)(L)ANTIGEN?' is valid since (W) is above (L) on the precedence list. However, '((THIN(W)LAYER)(L)PHOSPHOLIPID#)(A)LACTONE#' is not valid since (L) is below (A) on the precedence list. The only exception is the 'OR' operator. This operator may be used in combination with any other operator. For example, '(ATOMIC OR NUCLEAR)(W)REACTOR' is valid.

=> s (Valpha20 and Jalpha22)

L1 0 (VALPHA20 AND JALPHA22)

=> s tcr

L2 50709 TCR

=> s 12 (P) (Valpha20)

L3 1 L2 (P) (VALPHA20)

=> s 12 (P) (jalpha22)

L4 0 L2 (P) (JALPHA22)

=> s v20alpha (P) 12

L5 0 V20ALPHA (P) L2

=> s 12 (P) human

L6 12258 L2 (P) HUMAN

=> s alpha? (P) 16

L7 5019 ALPHA? (P) L6

=> s v20 (P) 17

L8 0 V20 (P) L7

=> s 20 (P) 17

L9 194 20 (P) L7

=> s 22 (P) 17

L10 109 22 (P) L7

=> s 19 or 110

L11 278 L9 OR L10

=> dup rem 111

PROCESSING COMPLETED FOR L11

L12 92 DUP REM L11 (186 DUPLICATES REMOVED)

=> dis l12 kwic 1-50

L12 ANSWER 18 OF 92 CAPIUS COPYRIGHT 2000 ACS DUPLICATE 15
AB . . . However, little is known about the nature of immune responses that might lead to tumor regression. We studied naturally arising human T-cell responses against RCC by combining mol. analyses of T-cell receptor (TCR) usage in primary tumors *in situ* with functional analyses of tumor-infiltrating lymphocytes (TILs) *in vitro*. TILs of patient 26 that were cultured *in vitro* showed a **human** leukocyte antigen (HLA-A*0201)-restricted cytotoxic activity specific for autologous tumor cells. These tumor-derived lymphocytes were dominated by a family of T cells expressing V.**alpha**.20- and V.**beta**.22-pos. **TCRs**. Their specificity-conferring third complementarity-detg. regions were highly homologous with respect to the loop length and selection of particular amino acids in both **TCR** chains. These characteristics are similar to those reported for antigen-selected murine T cells recognizing immunodominant epitopes of non-self proteins. To evaluate the biol. significance of these CTLs *in vivo*, we analyzed the corresponding **TCR** transcripts in the cryopreserved tumor material of patient 26 and in a second HLA-A*0201-pos. RCC patient whose tumor cells were also lysed by TIL-26. The *in situ* TIL **TCRs**, supporting the contention that immunodominant responses directed against a shared tumor-assocd. antigen occurred in both

L12 ANSWER 41 OF 92 MEDLINE

DUPLICATE 35

AB The T cell receptor (TCR) **alpha** beta variable (V) gene family usage of tumor-infiltrating lymphocytes (TIL) in different primary **human** malignant melanomas and corresponding metastatic lesions were characterized using a recently developed method using the reverse transcription coupled polymerase chain. . . histopathological samples of primary tumor material and demonstrated to be reproducible and to be useful for the assessment of V **alpha**- and V beta-gene family usage in tumor samples. The TIL in primary tumors were observed to preferentially express certain TCR V **alpha**- and V beta-gene families: V **alpha** 4, and V beta 8 were highly expressed in several of the primary tumors analyzed using this method. With respect to V **alpha** 22 and V beta 8, the preferential expression of these V-gene families was demonstrated to be due in situ clonal expansion. . . or V-D-J, respectively) corresponding to the RT-PCR products from one of the primary tumors. The observed preferential usage of certain TCR V **alpha** and V beta-genes strongly suggest the in situ clonal expansion of specific populations of T cells in accordance with recent. . . T cell populations probably react with certain melanoma-associated peptides presented by specific HLA molecules. The preferential usage of certain V **alpha**- and V beta-gene families observed in several tumors further supports the involvement of a limited number of shared melanocyte or. . . tumor material. In TIL in primary melanomas, a possible correlation was suggested between HLA-A2 and the preferential usage of the TCR V-gene families V **alpha** 4, V **alpha** 5, V **alpha** 22 and V beta 8, whereas the V beta 3-gene family appeared to be

L12 ANSWER 18 OF 92 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 15
AN 1998:484521 CAPLUS
DN 129:187626
TI Human renal cell carcinoma antigen-specific CTLs: antigen-driven selection
and long-term persistence *in vivo*
AU Jantzer, Petra; Schendel, Dolores J.
CS Institute of Immunology, University of Munich, Munich, 80336, Germany
SO Cancer Res. (1998), 58(14), 3078-3086
CODEN: CNREA8; ISSN: 0008-5472
PB American Association for Cancer Research
DT Journal
LA

L12 ANSWER 41 OF 92 MEDLINE
AN 96323429 . MEDLINE
DN 96323429
TI Analysis of T cell receptor alpha beta variability in tumor-infiltrating lymphocytes in primary and metastatic melanoma.
AU Zeuthen J; Birck A; Straten P T
CS Department of Tumor Cell Biology, Danish Cancer Society, Copenhagen, Denmark.
SO ARCHIVUM IMMUNOLOGIAE ET THERAPIAE EXPERIMENTALIS, (1995) 43 (2) 123-33.
Journal code: 790. ISSN: 0004-069X.
CY Poland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
E

DUPLICATE 35

L12 ANSWER 59 OF 92 MEDLINE
AN 93147728 MEDLINE

DUPLICATE 52

DN 93147728

TI Characterization of the T cell receptor repertoire causing collagen arthritis in mice.

AU Osman G E; Toda M; Kanagawa O; Hood L E

CS Division of Biology, California Institute of Technology, Pasadena 91125..

SO JOURNAL OF EXPERIMENTAL MEDICINE, (1993 Feb 1) 177 (2) 387-95.

Journal code: I2V. ISSN: 0022-1007.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

OS GENBANK-X67949

E

=> dis 112 18 38 41 59 abs

L12 ANSWER 18 OF 92 CAPLUS COPYRIGHT 2000 ACS DUPLICATE 15
AB Renal cell carcinomas (RCCs) are thought to be immunogenic, because cytokine-induced and even spontaneous tumor regression has been obsd. in a significant no. of patients. However, little is known about the nature of immune responses that might lead to tumor regression. We studied naturally arising **human** T-cell responses against RCC by combining mol. analyses of T-cell receptor (TCR) usage in primary tumors *in situ* with functional analyses of tumor-infiltrating lymphocytes (TILs) *in vitro*. TILs of patient 26 that were cultured *in vitro* showed a **human** leukocyte antigen (HLA-A*0201)-restricted cytotoxic activity specific for autologous tumor cells. These tumor-derived lymphocytes were dominated by a family of T cells expressing **V.alpha.20-** and **V.beta.22-pos. TCRs**.

Their specificity-conferring third complementarity-detg. regions were highly homologous with respect to the loop length and selection of particular amino acids in both TCR chains. These characteristics are similar to those reported for antigen-selected murine T cells recognizing immunodominant epitopes of non-self proteins. To evaluate the biol. significance of these CTLs *in vivo*, we analyzed the corresponding TCR transcripts in the cryopreserved tumor material of patient 26 and in a second HLA-A*0201-pos. RCC patient whose tumor cells were also lysed by TIL-26. The *in situ* TIL populations of both patients used related families of highly homologous TCRs, supporting the contention that immunodominant responses directed against a shared tumor-assocd. antigen occurred in both individuals *in vivo*. Furthermore, in the absence of overt metastatic disease, the tumor antigen-specific CTLs of patient 26 were shown to persist in the periphery 4 yr after removal of the primary tumor. These results demonstrate that antigen-driven T-cell responses specific for spontaneously arising carcinomas developed in these patients and showed long-term persistence, even in the absence of immunotherapy.

L12 ANSWER 38 OF 92 MEDLINE DUPLICATE 33
AB From the peripheral lymphocytes of a patient with Graves' disease, we established a T cell line using its reaction to a pool of 49 synthetic peptides corresponding to the entire **human** thyrotropin receptor (TSHR) sequence. This T cell line showed a specific response to the pool of peptides in a microproliferation assay (stimulation index: 4.8). Flow cytometry analysis revealed that the cell surface markers were CD4+ CD8-, T cell receptor (TcR) **alpha** beta+, and **Tcr** gamma delta-. To investigate T cell epitopes on TSHR, the T cell line reacted well against three groups: the N-terminal (amino acids 31-169) and C-terminal (338-420) regions of the extracellular domain and the N-terminal half (441-661) of the transmembrane domain of the receptor. This suggests a multiplicity of T cell epitopes on the TSHR, and was further supported by analysis of TcR gene expression in the cell line that showed the expression of 5 **V alpha** genes; **V alpha-1, 2, 10, 20, and w25**. In conclusion, the results of the present study indicated multiple T cell epitopes on the TSHR molecule including the transmembrane domain.

L12 ANSWER 41 OF 92 MEDLINE DUPLICATE 35
AB The T cell receptor (TCR) **alpha** beta variable (V) gene family usage of tumor-infiltrating lymphocytes (TIL) in different primary **human** malignant melanomas and corresponding metastatic lesions were characterized using a recently developed method using the reverse transcription coupled polymerase chain reaction (RT-PCR). This semiquantitative RT-PCR method could be adapted to analysis of formalin-fixed, paraffin-embedded histopathological samples of primary tumor material and demonstrated to be reproducible and to be useful for the assessment of **V alpha-** and **V beta**-gene family usage in tumor samples. The TIL in primary tumors were observed to preferentially express certain TCR **V alpha-** and **V beta**-gene families: **V alpha 4**, and **V beta 8** were highly expressed in several of the primary tumors analyzed using this method. With respect to **V alpha 22** and **V beta 8**, the preferential expression of these V-gene families was demonstrated to be due *in situ* clonal expansion of T cells by means of cloning and sequencing of the CDR3 regions (**V-J** or **V-D-J**, respectively) corresponding to the RT-PCR products from one of the primary

tumors. The observed preferential usage of certain **TCR V alpha** and **V beta**-genes strongly suggest the *in situ* clonal expansion of specific populations of T cells in accordance with recent results from others. These clonal T cell populations probably react with certain melanoma-associated peptides presented by specific HLA molecules. The preferential usage of certain **V alpha**- and **V beta**-gene families observed in several tumors further supports the involvement of a limited number of shared melanocyte or melanoma-associated peptides. Since the HLA status of the patients is obviously important to interpret these results, some of the patients were typed for HLA-A1 and -A2, the two most well-characterized restriction elements for melanoma-associated antigens, either serologically or by a newly developed RT-PCR method which similarly could be applied directly to the tumor material. In TIL in primary melanomas, a possible correlation was suggested between HLA-A2 and the preferential usage of the **TCR V-gene families V alpha 4, V alpha 5, V alpha 22 and V beta 8**, whereas the **V beta 3-gene family** appeared to be expressed together with HLA-A1. The **V-gene families** which were highly expressed in the primary tumors were generally not, or only very weakly, expressed in the corresponding metastases and vice versa, possibly reflecting a substantial change in the phenotype of the metastatic melanoma target cells. Continued studies of larger patient materials will be necessary to extend and validate these conclusions and of obvious interest for the further analysis of the T cell response in melanoma.

L12 ANSWER 59 OF 92 MEDLINE
 AB Collagen type II-induced arthritis (CIA) is generated in susceptible rodent strains by intradermal injections of homologous or heterologous native type II collagen in complete Freund's adjuvant. Symptoms of CIA are analogous to those of the **human** autoimmune disease, rheumatoid arthritis. CIA is a model system for T cell-mediated autoimmune disease. To study the T cell receptor (**TCR**) repertoire of bovine type II-specific T cells that may be involved in the pathogenesis of CIA in DBA/1Lac.J (H-2q) mice, 13 clonally distinct T cell hybridomas specific for bovine type II collagen have been established and the **alpha** and beta chains of their **TCRs** have been analyzed. These T cell hybridomas recognize epitopes that are shared by type II collagens from distinct species and not by type I collagens, and exhibit a highly restricted **TCR-alpha/beta repertoire**. The **alpha** chains of the **TCRs** employ three **V alpha** gene subfamilies (**V alpha 11, V alpha 8, and V alpha 22**) and four **J alpha** gene segments (**J alpha 42, J alpha 24, J alpha 37, and J alpha 32**). The **V alpha 22** is a newly identified subfamily consisting of approximately four to six members, and exhibits a high degree of polymorphism among four mouse strains of distinct **V alpha** haplotypes. In addition, the beta chains of the **TCRs** employ three **V beta** gene subfamilies (**V beta 8, V beta 1, and V beta 6**), however the **V beta 8.2** gene segment is preferentially utilized (58.3%). In contrast, the **J beta** gene segment usage is more heterogeneous. On the basis of the highly limited **TCR-alpha/beta** repertoire of the **TCRs** of the panel of bovine type II-specific T cell hybrid clones, a significant reduction (60%) of the incidence of arthritis in DBA/1Lac.J mice is accomplished by the use of anti-**V beta 8.2** antibody therapy.

=> logoff

ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
 LOGOFF? (Y)/N/HOLD:Y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
FULL ESTIMATED COST	ENTRY	SESSION
	90.13	90.43
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE	ENTRY	SESSION
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WEST Search History

DATE: Saturday, June 08, 2002

Set Name Query

side by side

DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=OR

L6 @ad<19960624 and 15

19 L6

L5 L4 and AD<19960624

86 L5

L4 L3 and alpha

86 L4

L3 ((kidney or renal) same (carcinoma\$4 or neoplas\$6 or tumor\$))and cdr3

100 L3

L2 L1 and cdr3

6 L2

L1 (schendel)[IN]

164 L1

END OF SEARCH HISTORY

L4 ANSWER 1 OF 15 CAPLUS COPYRIGHT 2001 ACS
 ACCESSION NUMBER: 2000:871678 CAPLUS
 DOCUMENT NUMBER: 134:176980
 TITLE: Expression of B7.1 (CD80) in a renal cell carcinoma line allows expansion of tumor-associated cytotoxic T lymphocytes in the presence of an alloresponse
 AUTHOR(S): Schendel, D. J.; Frankenberger, B.; Jantzer, P.; Cayeux, S.; Nossner, E.; Willimsky, G.; Maget, B.; Pohla, H.; Blankenstein, T.
 CORPORATE SOURCE: Institute of Molecular Immunology, GSF National Research Center for the Environment and Health, Munich, Germany
 SOURCE: Gene Ther., (2000), 7(23), 2007-2014
 PUBLISHER: Nature Publishing Group
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The authors have selected a well-characterized human renal cell carcinoma (RCC) line as the basis for development of a genetically engineered tumor cell vaccine to be applied in an allogeneic setting. This cell line was genetically modified by retroviral transduction to express B7.1 costimulatory mols. The unmodified tumor cells and B7.1-expressing tumor cells were compared for their ability to induce tumor-assoc. responses in allogeneic peripheral blood mononuclear cells (PBMC) of two normal control donors having single MHC class I allele matches with the tumor cells. PBMC primed using B7.1-modified tumor cells showed a preponderance of CD3+CD8+ cytotoxic T lymphocytes (CTL) that proliferated over extended periods of time in mixed lymphocyte tumor cell (MLTC) cultures. Strong cytolytic activity developed in the primed populations and included alloreactive CTL with specificity for mismatched HLA-A, -B and -C mols. Nevertheless, it was possible to isolate CTL clones that were able to lyse tumor cells but not lymphoblastoid cells that expressed all the corresponding alloreactivities. Thus, induction of complex alloreactive responses did not hinder the development of tumor-assoc. CTL *in vitro*. These results support the use of this genetically modified allogeneic tumor cell line for vaccination of partial-MHC matched RCC patients.

REFERENCE COUNT: 40

REFERENCE(S):
 (1) Antonia, S; Cancer Res 1995, V55, P2253 CAPLUS
 (2) Bain, C; Int J Cancer 1996, V67, P769 CAPLUS
 (3) Boon, T; Immunol Today 1997, V18, P267 CAPLUS
 (4) Chen, L; Cell 1992, V71, P1093 CAPLUS
 (5) Daniel, P; J Immunol 1997, V159, P3808 CAPLUS

AU Schendel, D. J.; Frankenberger, B.; Jantzer, P.; Cayeux, S.; Nossner, E.; Willimsky, G.; Maget, B.; Pohla, H.; Blankenstein, T.

L4 ANSWER 2 OF 15 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1999:667840 CAPLUS
 DOCUMENT NUMBER: 131:296206
 TITLE: Method for the preparation of a polycistronic T-cell receptor-expression cassette and its insertion into human T-cells

INVENTOR(S): Schendel, Dolores; Jantzer, Petra
 PATENT ASSIGNEE(S): Germany

SOURCE: Ger. Offen., 20 pp.
 DOCUMENT TYPE: Patent
 LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19816129	A1	1999104	DE 1998-19816129	19980409
WO 9952943	A1	1999102	WO 1999-EP2171	19990330
W: JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1068237	A1	20010117	EP 1999-917915	19990330
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, NL, SE, PT, IE, FI				
PRIORITY APPLN. INFO.:			DE 1998-19816129	19980409
			WO 1999-EP2171	19990330

X

AB The invention concerns a method for the prepn. of a polycistronic expression cassette and the prodn. of the T-cell receptors in human T-cell lines by using a plasmid vector that codes at least fragments of the C-regions of the TCR.α. and TCR.β. chains along with 5'-restriction sites. The method allows the generation of novel "artificial T cells" with a defined T-cell receptor subtype. A polycistronic expression cassette using an internal ribosome entry sequence (IRES) is described.

IN Schendel, Dolores; Jantzer, Petra

L4 ANSWER 3 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1999:184299 BIOSIS
 DOCUMENT NUMBER: PREV199900184299
 TITLE: MHC class I restricted tumor cell lysis in renal cell carcinoma

AUTHOR(S): Oberneder, Ralph; Jantzer, Petra; Nossner, Elfriede; Hofstetter, Alfons; Schendel, Dolores J.
 CORPORATE SOURCE: Munich, Germany
 SOURCE: Journal of Urology, (April, 1999), Vol. 161, No. 4 SUPPL., pp. 144-145

Meeting Info.: 94th Annual Meeting of the American Urological Association, Inc. Dallas, Texas, USA May 1-6, 1999 American Urological Association
 ISSN: 0022-5347.

DOCUMENT TYPE: Conference
 LANGUAGE: English
 AU Oberneder, Ralph; Jantzer, Petra; Nossner, Elfriede; Hofstetter, Alfons; Schendel, Dolores J.

L4 ANSWER 4 OF 15 MEDLINE
 ACCESSION NUMBER: 1998343568 MEDLINE
 DOCUMENT NUMBER: 98343568

DUPLICATE 2

TITLE: Human renal cell carcinoma/antigen-specific CTLs: antigen-driven selection and long-term persistence *in vivo*.
 AUTHOR: Jantzer, P; Schendel, D.J.
 CORPORATE SOURCE: Institute of Immunology, University of Munich, Germany.
 SOURCE: CANCER RESEARCH, (1998) Jul 15; 58 (14) 3078-86.
 PUB. COUNTRY: United States
 Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199810
ENTRY WEEK: 19981003

AB Renal cell carcinomas (RCCs) are thought to be immunogenic, because cytokine-induced and even spontaneous tumor regression has been observed in a significant number of patients. However, little is known about the nature of immune responses that might lead to tumor regression. We studied naturally arising human T-cell responses against RCC by combining molecular analyses of T-cell receptor (TCR) usage in primary tumors *in situ* with functional analyses of tumor-infiltrating lymphocytes (TILs) *in vitro*. TILs of patient 26 that were cultured *in vitro* showed a human leukocyte antigen (HLA-A*0201)-restricted cytotoxic activity specific for autologous tumor cells. These tumor-derived lymphocytes were dominated by a family of T cells expressing V alpha20- and V beta22-positive TCRs. Their specificity-conferring third complementarity-determining regions were highly homologous with respect to the loop length and selection of particular amino acids in both TCR chains. These characteristics are similar to those reported for antigen-selected murine T cells recognizing immunodominant epitopes of non-self proteins. To evaluate the biological significance of these CTLs *in vivo*, we analyzed the corresponding TCR transcripts in the cryopreserved tumor material of patient 26 and in a second HLA-A*0201-positive RCC patient whose tumor cells were also lysed by TIL-26. The *in situ* TIL populations of both patients used related families of highly homologous TCRs, supporting the contention that immunodominant responses directed against a shared tumor-associated antigen occurred in both individuals *in vivo*. Furthermore, in the absence of overt metastatic disease, the tumor antigen-specific CTLs of patient 26 were shown to persist in the periphery 4 years after removal of the primary tumor. These results demonstrate that antigen-driven T-cell responses specific for spontaneously arising carcinomas developed in these patients and showed long-term persistence, even in the absence of immunotherapy.

AU Jantzer P; Schendel D J

L4 ANSWER 5 OF 15 MEDLINE
ACCESSION NUMBER: 97375563 MEDLINE
DOCUMENT NUMBER: 97375563

DUPLICATE 3

TITLE: Cellular and molecular analyses of major histocompatibility complex (MHC) restricted and non-MHC-restricted effector cells recognizing renal cell carcinomas: problems and perspectives for immunotherapy.

AUTHOR: Schendel D J; Oberneder R; Falk C S; Jantzer P; Kressenstein S; Maget B; Hofstetter A; Riethmuller G; Nossner E

CORPORATE SOURCE: Institut für Immunologie, Ludwig-Maximilians-Universität München, Munich, Germany.

SOURCE: JOURNAL OF MOLECULAR MEDICINE, (1997 Jun) 75 (6) 400-13.

Ref: 119
Journal code: B8C. ISSN: 0946-2718
GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY WEEK: 19971102

AB Renal cell carcinomas belong to the small group of tumors that are able to induce antitumor responses. Here we describe two general types of cytotoxic effector lymphocytes that can eliminate autologous tumor cells and discuss the role that major histocompatibility complex encoded molecules play in governing their specificities. Improved understanding of the cellular and molecular basis of renal cell carcinoma recognition opens new avenues of research with the potential to develop better immunotherapies for patients with metastatic disease.

AU Schendel D J; Oberneder R; Falk C S; Jantzer P; Kressenstein S; Maget B; Hofstetter A; Riethmuller G; Nossner E

L4 ANSWER 6 OF 15 MEDLINE

ACCESSION NUMBER: 97187412 MEDLINE

DUPLICATE 4

DOCUMENT NUMBER: 97187412

TITLE: The HLA likes and dislikes of allospecific and non-MHC-restricted cytotoxic T lymphocytes.

AUTHOR: Nossner E; Falk C S; Jantzer P; Reinhardt C; Steinle A; Schendel D J

CORPORATE SOURCE: Institute of Immunology, University of Munich, Germany.
SOURCE: IMMUNOLOGICAL REVIEWS, (1996 Dec) 154 105-35. Ref: 131

PUB. COUNTRY: Denmark
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199707
ENTRY WEEK: 19970703

AU Nossner E; Falk C S; Jantzer P; Reinhardt C; Steinle A;
Schendel D J

L4 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1997:133304 BIOSIS

DOCUMENT NUMBER: PREV199799432507

TITLE: The HLA likes and dislikes of allospecific and non-MHC-restricted cytotoxic T lymphocytes.

AUTHOR(S): Noessner, Elfriede; Falk, Christine S.; Jantzer, Petra; Reinhardt, Carsten; Steinle, Alexander;

CORPORATE SOURCE: (1) Inst. Immunol., Univ. Munich, Goethestr. 31, 80336
Munich Germany
SOURCE: Immunological Reviews, (1996) Vol. 0, No. 154, pp. 105-135.
ISSN: 0105-2896.

DOCUMENT TYPE: General Review

LANGUAGE: English

AU Noessner, Elfriede; Falk, Christine S.; Jantzer, Petra;
Reinhardt, Carsten; Steinle, Alexander; Schendel, Dolores J. (1)

L4 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

ACCESSION NUMBER: 1995:384452 BIOSIS

DOCUMENT NUMBER: PREV199598398752

TITLE: In vivo abundance of HLA-B35 alloreactive T cells with homologous TCR.

AUTHOR(S): Steinle, A.; Reinhardt, C.; Jantzer, P.; Seebart,

CORPORATE SOURCE: K.; Schendel, D. J.
SOURCE: Univ. Munich, Munich Germany
9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY.. (1995) pp. 649.
The 9th International Congress of Immunology.
Publisher: 9th International Congress of Immunology San
Francisco, California, USA.
Meeting Info.: Meeting Sponsored by the American
Association of Immunologists and the International Union of
Immunological Societies San Francisco, California, USA July
23-29, 1995
DOCUMENT TYPE: Conference
LANGUAGE: English
AU Steinle, A.; Reinhardt, C.; Jantzer, P.; Seebart, K.;
Schendel, D. J.

L4 ANSWER 9 OF 15 MEDLINE
ACCESSION NUMBER: 95138683 MEDLINE
DOCUMENT NUMBER: 95138683
TITLE: In vivo expansion of HLA-B35 alloreactive T cells sharing
homologous T cell receptors: evidence for maintenance of an
oligoclonally dominated allo specificity by persistent
stimulation with an autologous MHC/peptide complex.
AUTHOR: Steinle A; Reinhardt C; Jantzer P; Schendel D J
CORPORATE SOURCE: Institute of Immunology, University of Munich, Germany..
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1995 Feb 1) 181 (2)
503-13.
PUB. COUNTRY: Journal code: 12V. ISSN: 0022-1007.
United States
LANGUAGE: Journal; Article; (JOURNAL ARTICLE)
FILE SEGMENT: English
OTHER SOURCE: Priority Journals; Cancer Journals
ENTRY MONTH: GENBANK-Z46961; GENBANK-Z46963; GENBANK-Z46962
199505

AB The nature of alloantigens seen by T lymphocytes, in particular the role
of peptides in allorecognition, has been studied intensively whereas
knowledge about the in vivo emergence, diversity, and the structural basis
of specificity of alloreactive T cells is very limited. Here we describe
human T cell clones that recognize HLA-B35 alloantigens in a
peptide-dependent manner. TCR sequence analysis revealed that several of
these allospecific clones utilize homologous TCR: they all express
TCRA2S3J36C1 and TCRBV4S1J2S7C2 chains with highly related CDR3
sequences. Thus peptide-specific alloreactivity is reflected in homologous
CDR3 sequences in a manner similar to that described for T cells that
recognize nominal peptide/self-MHC complexes. The in vivo frequency of
this TCR specificity was studied in unstimulated PBL of the responding
cell donor who was not sensitized against HLA-B35. The vast majority
(approximately 75%) of the VA2S3J36 junctional regions obtained from two
samples of PBL, isolated at a 9-yr interval, encode CDR3 identical or
homologous to those of the functionally characterized HLA-B35 allospecific
T cells. These data are most easily explained by a model of alloreactivity
in which persistent or recurrent exposure to a foreign peptide/self-MHC
complex led to the in vivo expansion and long-term maintenance of specific
T cells that show fortuitous crossrecognition of an HLA-B35/peptide
complex and dominate the alloresponse against HLA-B35.

AU Steinle A; Reinhardt C; Jantzer P; Schendel D J

L4 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1995:326448 BIOSIS
DOCUMENT NUMBER: PREV199598340748
TITLE: Long-term in vivo expansion of HLA-B35 alloreactive T cells
with homologous TCR suggests crosstimulation via a
persistent peptide/self MHC complex.
AUTHOR(S): Steinle, Alexander; Reinhardt, Carsten; Jantzer,
Petra; Schendel, Dolores J.
CORPORATE SOURCE: Inst. Immunol., Univ. Munich, 80336 Muenchen Germany
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0,
No. 21A, pp. 177.
Meeting Info.: Keystone Symposium on Control and
Manipulation of the Immune Response Taos, New Mexico, USA
March 16-22, 1995
ISSN: 0733-1959.

DOCUMENT TYPE: Conference
LANGUAGE: English
AU Steinle, Alexander; Reinhardt, Carsten; Jantzer, Petra;
Schendel, Dolores J.

L4 ANSWER 11 OF 15 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:74021 CAPLUS
DOCUMENT NUMBER: 124:143403
TITLE: Recruitment of MHC-restricted cytotoxic T lymphocytes
specific for renal cell carcinoma to the tumor in situ
AUTHOR(S): Jantzer, Petra; Oberneder, Ralph; Maget,
Barbara; Schendel, Dolores J.
CORPORATE SOURCE: Institut für Immunologie, Ludwigs-Maximilians-
Universität, Munich, Germany
SOURCE: Biol. Renal Cell Carcinoma, [Proc. Symp.], 3rd (1995),
Meeting Date 1994, 84-93. Editor(s): Bukowski, Ronald
M.; Finke, James H.; Klein, Eric A. Springer: New
York, N. Y.
CODEN: 62GUAA
DOCUMENT TYPE: Conference
LANGUAGE: English

AB Lymphocytic populations from a patient with renal cell carcinoma (RCC)
were characterized. Tumor infiltrating lymphocytes (TIL) cultured with
low amts. of rIL-2 displayed the classical phenotype of cytotoxic T cells
and were highly specific for autologous tumor cells. In situ TIL were
limited in their TCR heterogeneity. Evidence was obtained for specific
recruitment of MHC-restricted cytotoxic T cells to the tumor site.
AU Jantzer, Petra; Oberneder, Ralph; Maget, Barbara; Schendel,
Dolores J.

L4 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1994:338156 BIOSIS
DOCUMENT NUMBER: PREV199497351156
TITLE: Fine specificity analysis of MHC-restriction and T-cell
receptor usage of tumor infiltrating lymphocytes
recognizing autologous and allogeneic renal cell
carcinomas.
AUTHOR(S): Schendel, Dolores J; Jantzer, Petra;
Kressenstein, Susanne; Maget, Barbara; Oberneder, Ralph;
Seebart, Kimberly; Steinle, Alexander
CORPORATE SOURCE: Munich Germany

SOURCE: Journal of Urology, (1994), 151, No. 5 SUPPL., pp.
 484A.
 Meeting Info.: Eighty-ninth Annual Meeting of the American
 Urological Association San Francisco, California, USA May
 14-19, 1994
 ISSN: 0022-5347.

DOCUMENT TYPE: Conference
 LANGUAGE: English
 AU Schendel, Dolores J.; Jantzer, Petra; Kressenstein,
 Susanne; Maget, Barbara; Oberneder, Ralph; Seebart, Kimberly; Steinle,
 Alexander

L4 ANSWER 13 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
 ACCESSION NUMBER: 1995:47085 BIOSIS
 DOCUMENT NUMBER: PREV199598061385
 TITLE: Identification and characterization of highly specific
 tumor infiltrating lymphocytes in a primary renal cell
 carcinoma.

AUTHOR(S): Jantzer, P.; Schendel, D. J.
 CORPORATE SOURCE: Institut Immunologie, LMU Muenchen, Munich Germany
 SOURCE: Immunobiology, (1994) Vol. 191, No. 2-3, pp. 212-213.
 Meeting Info.: XXVth Meeting of the Society of Immunology
 Konstanz, Germany September 21-24, 1994
 ISSN: 0171-2985.

DOCUMENT TYPE: Conference
 LANGUAGE: English
 AU Jantzer, P.; Schendel, D. J.

L4 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
 ACCESSION NUMBER: 1995:46995 BIOSIS
 DOCUMENT NUMBER: PREV199598061295
 TITLE: Long term *in vivo* expansion of HLA-B35 alloreactive T cells
 with homologous TCRs suggests cross-stimulation via a
 persistent peptide/self MHC complex.

AUTHOR(S): Steinle, A.; Reinhardt, C.; Jantzer, P.;
 Schendel, D. J.
 CORPORATE SOURCE: Inst. Immunol., Univ. Muenchen, Muenchen Germany
 SOURCE: Immunobiology, (1994) Vol. 191, No. 2-3, pp. 155.
 Meeting Info.: XXVth Meeting of the Society of Immunology
 Konstanz, Germany September 21-24, 1994
 ISSN: 0171-2985.

DOCUMENT TYPE: Conference
 LANGUAGE: English
 AU Steinle, A.; Reinhardt, C.; Jantzer, P.; Schendel, D. J.

L4 ANSWER 15 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
 ACCESSION NUMBER: 1994:46487 BIOSIS
 DOCUMENT NUMBER: PREV199497059487
 TITLE: T cell receptor repertoire of tumor-infiltrating
 lymphocytes (TIL) in renal cell carcinoma (RCC).

AUTHOR(S): Jantzer, P.; Segurado, O. G.; Schendel, D.
 J.
 CORPORATE SOURCE: Inst. Immunol., Univ. Munich, Munich Germany
 SOURCE: Immunobiology, (1993) Vol. 189, No. 1-2, pp. 156.
 Meeting Info.: 24th Meeting of the Society for Immunology
 Leipzig, Germany September 30-October 2, 1993
 ISSN: 0171-2985.

DOCUMENT TYPE: Conference
 LANGUAGE: English
 AU Jantzer, P.; Segurado, O. G.; Schendel, D. J.

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